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October 26 - brain damage part 2, parkinsons’s disease, alzheimer’s disease, traumatic brain

Physiological Psychology (University of Saskatchewan)

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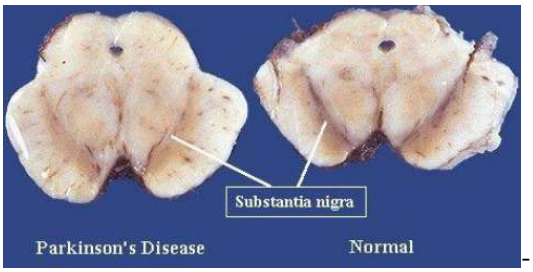
October 26, 2021

CHAPTER 10

LECTURE 16: BRAIN DAMAGE PART 2

PARKINSONS'S DISEASE

- Shaking Palsy – due to tremors
- Occurs in nigrostriatal pathway, originating from cell death in substantia nigra (in midbrain)
- Dopamine is affected in basal ganglia
    - Reduced dopamine -> Parkinson's
- Basal ganglia – allow us to fine tune movements; suppress movement we don't want to do
- Loss of dopaminergic neurons in substantia nigra
- Types of symptoms
    - Hyperkinetic – unwanted movements; shaking
    - Hypokinetic - unable to initiate/make the movements we want
- 

- normal – black/dark shade or colour
- Occurs in about 1% of population over the age 55
    - More common in males
- Symptoms
    - Disorder of posture - slouch
    - Akinesia – inability to initiate movement
    - Dyskinesia – hyperkinetic symptoms
    - Mask-like affect – hard to control facial muscles; can't express emotions well
- Treatment in the first half of 20th century was neurosurgery – not very successful
- 2 major discoveries
    - Dopamine the major neurotransmitter in basal ganglia (1950s)
    - Synthetic dopamine agent *Levodopa* (1960s) (aka L-dopa) – a miracle drug
- Levodopa (L-dopa) – primary treatment
    - Taken in conjunction with carbidopa (prevent L-dopa from being broken down before it gets to the brain; allow L-dopa to be more efficiently processed in the brain; help reduce tolerance)
    - Can result in long-term tolerance
    - Continued use -> tolerance to the drug -> drug is less effective
        - They need to get off the drug to diminish tolerance
- A rodent model of Parkinson's uses MPTP
    - MPTP – mimics the effects of Parkinson's disease; and does it quickly
        - Rapidly kills dopaminergic neurons in the substantia nigra (within hours)

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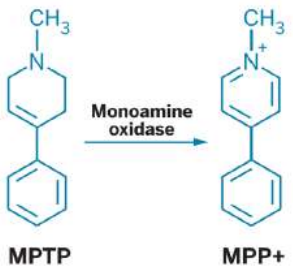
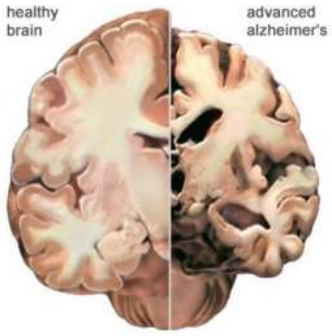
- “Frozen Addicts” (took the drugs -> developed Parkinson’s) and discovery of MPTP (is not neurotoxic, but will interact with monoamine oxidase, an enzyme, that will make it neurotoxic -> kills dopamine neurons in midbrain)
  - 

- Search for alternative therapies
  - Deep brain stimulation – tiny electrode is implanted deep in midbrain to generate electrical signals that are not being sent; to replace the signalling
    - Tiny electrode implanted deep in the midbrain
    - Connected to pacemaker (powers the electrodes)
    - Electrical signals substitute those depleted by neuron loss
    - Effective, but invasive (brain surgery)
    - Electrode can move slightly -> need to be readjusted

ALZHEIMER’S DISEASE

- Cause is unknown, but there is a genetic link
  - Carrying the gene – risk factor
- Stronger maternal link – risk factor if mother has Alzheimer’s
- Typically, later 60s and 70s
- Early-onset type – mid to late 40s; has a different genetic link; a different gene is involved
- Beta-amyloid plaques
  - Build up in the brain and suffocate neurons
- Neurofibrillary tangles – co-occur with plaques
  - String-like filaments associated with plaque buildup and cell death
- Post-mortem cell loss is extensive
  - 

  - Not easy to definitively diagnosed when they’re alive
  - Easy to diagnose when dead already
  - Can become a global disease of the brain
- Current treatment options are limited

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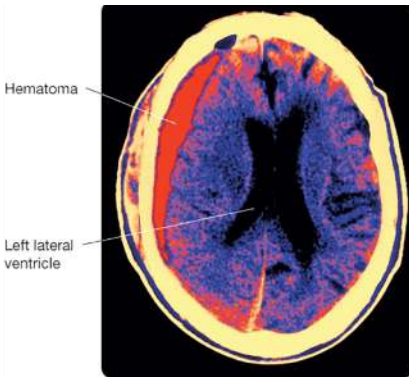
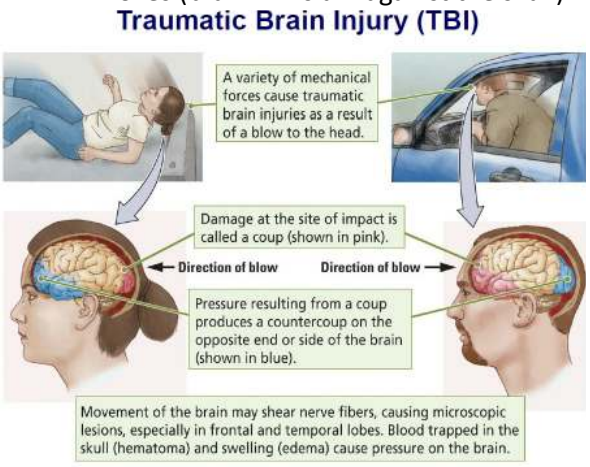
    - Acetylcholine agonists – main neurotransmitter
        - Block the breakdown of acetylcholine
        - Can limit advance of disease, but can't prevent it from progression
- Focus on early diagnosis
    - PET and MRI – use early preventive techniques
- Transgenic mouse models
    - Block production of beta-amyloid plaques

TRAUMATIC BRAIN INJURY (TBI)

- Contusion – can still occur even without outside damage
    - 

    - Results in hematoma (build up of blood in skull) – can exert pressure in the brain; need surgery to remove it
    - Brain hitting the skull
    - Countercoup – hit on left side of the head -> bleeding in right side of head since brain moves (brain will slam against the skull) -> damage to both sides of head
- 
Traumatic Brain Injury (TBI)
A variety of mechanical forces cause traumatic brain injuries as a result of a blow to the head.
Damage at the site of impact is called a coup (shown in pink).
Direction of blow
Direction of blow
Pressure resulting from a coup produces a countercoup on the opposite end or side of the brain (shown in blue).
Movement of the brain may shear nerve fibers, causing microscopic lesions, especially in frontal and temporal lobes. Blood trapped in the skull (hematoma) and swelling (edema) cause pressure on the brain.
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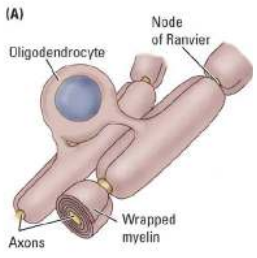
- Chronic traumatic encephalopathy (CTE)
    - Thought to result from chronic concussions -> learning and behavioural problems
    - E.g., boxing, football

RECOVERY FROM TRAUMATIC BRAIN INJURY

- Recover may continue for 2-3 years and longer
    - Most cognitive recovery occurs in the first 6-9 months
    - Poor attention and memory functions
- Increased risk for developing future neurodegenerative disorder
    - Dementia and Alzheimer's

MULTIPLE SCLEROSIS (MS)

- A neurodegenerative disorder
- Loss of myelin in the CNS
    - Myelin sheath and in some cases the axons are destroyed
        - Myelin is broken down or destroyed
    - Severe motor deficits – can lead to paralysis
- An autoimmune disease
    - Immune system fails to discriminate between foreign pathogens in the body and the body itself
        - Protein sequences of foreign microbes may be similar to structures found in myelin
    - Immune system will attack the myelin, thinking that it's a foreign pathogen – immune system will attack itself
    - Loss of myelin -> neuro signalling deficit
- Symptoms
    - Ataxia – hard to pick up and move objects
    - Numbness – lack of feeling
    - Poor vision
        - One or both eyes
        - Pain on parts of body
    - Periods of partial recovery – no symptoms; like on remission
- Men and women – 3x more prevalent in women
- Race and geography – more common in Caucasians and northern latitudes; may be related to reduced sunlight
- 

NEURAL REGENERATION IN HUMANS

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- Only occurs in peripheral nervous system
    - Neural regeneration can occur in the mammalian PNS
    - Facilitated by Schwann cells
        - Schwann cells – form the myelin in neurons in PNS
        - Damage in PNS -> schwann cells will promote neurotrophic factors (will aid and repair cells) -> will regrow and re-establish connections to other neurons by promotion of cell Adhesion Molecules (chemical signals that allows axons to establish new synaptic targets)

NEURAL REGENERATION IN HUMANS

- The less damage to schwann cells, the more successful the regeneration
- Cells can regenerate as long as the schwann cell aren't that badly damaged
- The environment of the PNS is one that promotes regeneration
    - The CNS inhibits axonal regrowth
        - Demyelination of spared axons
    - Brain and spinal injury -> severed axons -> demyelination of axons
        - Injury to brain and spinal cord -> limited options on how to repair it

RECOVERY OF FUNCTION AFTER CNS DAMAGE

- Plasticity of existing connections (more dendrites can be established/reconnect) in the brain can recover some degree of function
    - Increased dendritic branching
    - Stroke and TBI
    - Re-establish functions via therapy
- Promise of stem cell transplantation
    - The opportunity of repopulating areas of CNS with new cells
    - Stem cell – undifferentiated cells; may not always behave properly
- Problems to everyone
    - Long-term survival of stem-cell derived neurons
    - Unpredictable interactions with existing cells
    - Risk of tumors for cells failing to differentiate properly

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